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Oral Sessions

Angiotensin II-induced constriction via Rho kinase activation in pressure-overloaded rat thoracic aortas

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Inactivation of myosin light chain phosphatase through myosin phosphatase targeting subunit 1 (MYPT1) phosphorylation by Rho kinase is important for angiotensin II (Ang II)-induced constriction. However, the mechanism of Rho kinase activation by Ang II is unknown. We investigated whether Ang II-induced constriction in pressure-overloaded rat thoracic aortas is mediated by Rho kinase activation through Src, epidermal growth factor receptor (EGFR), extracellular signal-regulated kinase (Erk), and janus kinase (JAK).

The pressure-overload in rat thoracic aortas was produced by suprarenal abdominal aortic coarctation. After 4 weeks, thoracic aortas were excised and performed organ chamber experiments. Protein levels were measured by Western blotting.

Contractile response to Ang II significantly attenuated by inhibitors of Rho kinase, Erk1/2, Src, and EGFR in sham-treated and pressure-overloaded rats. Total and phosphorylated levels of MYPT1 and Src were increased in pressure-overloaded rat thoracic aortas. These data suggest that Ang II-induced constriction is mediated by Rho kinase activation via Src, EGFR, and Erk in pressure-overloaded rat thoracic aortas.